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# **EXHIBIT B**



## (12) United States Patent

### Petzelbauer

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#### (54) THERAPEUTIC FIBRIN-DERIVED PEPTIDES AND USES THEREOF

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#### **ABSTRACT**

The invention relates to peptides having the general formula (I), or a salt or amide thereof, wherein  $R_1$  and  $R_2$  are either the same or different, wherein R, and R<sub>2</sub> are each selected from the group consisting of hydrogen and a saturated or unsaturated hydrocarbon residue, said residue having from 1 to 10 carbon atoms, wherein Z<sub>1</sub> is selected from the group consisting of histidine and proline, wherein Z<sub>2</sub> is selected from the group consisting of an arginine and a peptide comprising an initial arginine and having from 2 to 30 amino acids. The invention also relates to methods using the peptides of the present invention in the treatment of inflammation.

3 Claims, No Drawings

### THERAPEUTIC FIBRIN-DERIVED PEPTIDES AND USES THEREOF

#### CROSS REFERENCE TO RELATED APPLICATIONS

The present application is a continuation of U.S. patent application Ser. No. 10/459,030, filed Jun. 11, 2003, now U.S. Pat. No. 7,271,144, which is a continuation of International Patent Application No. PCT/AT01/00387, filed Dec. 7, 2001, 10 published in German on Jun. 20, 2002 as International Patent Publication No. WO02/248180, which claims priority to Austrian Application No. AT A 2063/2000, filed Dec. 12, 2000, all of which are incorporated in their entireties herein.

#### **BACKGROUND**

The invention concerns peptides and/or proteins, their use for preparing a therapeutic and/or preventive pharmaceutical composition as well as a pharmaceutical composition.

Substances for the inhibition or prevention of inflammatory reactions, so-called immunosuppressants, which so far have been used for prophylaxis and therapy, generally comprise two distinct groups. Firstly, derivatives of a hormone, i.e. cortisone, naturally occurring in the body, and secondly, exogenous immunosuppressants such as cyclosporin and its derivatives, azathioprine, cyclophosphamide etc. All those substances possess anti-inflammatory effects but they show substantial side reactions in long-term therapy. Those side reactions have a limiting effect on long-term therapy, which is why those substances are used alternately or in combination in order to keep side effects on a tolerable level or in order to be able to actually proceed with the therapy. As examples of side reactions, the pathological fractures associated with cortisone are to be mentioned, which fractures are caused by the osteoporotic effect of the cortisone, or the renal failure which may be caused by cyclosporin. Those side reactions are inevitable with both groups of compounds, and hence it is merely a question of the duration of the therapy and of the total dose at what point the therapy must be stopped.

#### SUMMARY OF THE INVENTION

The present invention has as its object to provide new pharmaceutical products which are suitable for preventing or inhibiting inflammatory effects and which only show minor 45 side effects. A further object consists in providing long-term therapy.

In the following, the amino acids of the peptides according to the invention are referred to by the usual abbreviations, which denote the  $\alpha$ -amino acids.

By "analogues," a peptide is understood which, by derivatisation, substitution, preferably homologous substitution, deletion and/or insertion, is derived from the sequence of the fibrin and in particular from the preferred sequences.

The peptides or protein according to the invention exhibit 55 the general formula I

$$\begin{array}{c|c} R_1 & H & O \\ N - C & H & C \\ R_2 & H & C \end{array}$$

wherein R<sub>1</sub> and R<sub>2</sub>, being equal or different, denote hydrogen, 65 a saturated or unsaturated hydrocarbon residue comprising from 1 to 3, in particular up to 10, carbon atoms,

Z<sub>1</sub> denotes a histidine or proline residue,

Z<sub>2</sub> denotes an arginine residue, a peptide residue or a protein residue comprising an initial arginine residue, in particular comprising from 2 to 30 amino acids, as well as the salts thereof, and, f.i., also amides, or mixtures with each other and /or with at least one further substance for therapeutic and/or preventive use in human and/or veterinary medicine, whereby in particular only L-amino acids are provided. Sequences of formula I are listed in Table 1.

It was completely surprising that the specified amino acid sequence prevents the adhesion of cells from the bloodstream to endothelial cells of the vascular wall and/or their subsequent transmigration from the blood into the tissue.

The peptides or protein according to the invention exhibit the general formula II

25 wherein R<sub>1</sub> and R<sub>2</sub>, being equal or different, denote hydrogen, a saturated or unsaturated hydrocarbon residue comprising from 1 to 3, in particular up to 10, carbon atoms,

Z<sub>1</sub> denotes a histidine or proline residue,

Arg denotes an arginine,

Z<sub>3</sub> denotes a proline or valine residue,

Z<sub>4</sub> denotes a leucine or valine residue,

Z<sub>5</sub> denotes a protein residue or a peptide residue, in particular comprising from 2 to 30 amino acids, or an alcohol comprising from 1 to 3, in particular up to 10, carbon atoms, or an organic or inorganic base residue, as well as the salts thereof, and, f.i., also amides, or mixtures with each other and/or with at least one further substance for therapeutic and/or preventive use in human and/or veterinary medicine, whereby in particular only L-amino acids are provided. Sequences of formula II are listed in Table 2.

It was completely surprising that parts of the sequence, peptides or fragments of the fibrinogen exhibit anti-inflammatory effects. Without being bound by such theoretical considerations, said effects might be based on the fact that the fibrin binds to endothelial cells via its neo-N-terminus of the Bbeta-chain and to cells in the bloodstream via the sequence of the Aalpha-chain, thereby leading to the adhesion and transmigration of cells into the tissue. Those bindings exhibit 50 a side reaction in that the formation of fibrin is inhibited. However, said inhibition does not constitute a potential disadvantage to the patient since the blood coagulation is sufficient also in the absence of fibrin if slight injuries occur. Only in case of surgical treatment, it might optionally be suitable to stop such kind of therapy. Other side reactions may substantially be ruled out, since those substances only interact with natural ligands. Furthermore, the natural defense is not affected adversely by the leukocytes in the blood. Thus, the composition of the same, such as granulocytes, lymphocytes and monocytes, remains unaffected so that the natural defense process is maintained and the defense against infections in the blood remains unchanged.

### DETAILED DESCRIPTION

Fibrinogen is produced in the liver and, in this form, is biologically inactive and normally is provided in the blood at

concentrations of around 3 g/l. By proteolytic cleavage of the proenzyme prothrombin, thrombin is formed which cleaves off the fibrinopeptides A and B from the fibrinogen. In doing so, fibringen is transformed into its biologically active form. Fibrin and fibrin cleavage products are generated.

Thrombin is formed during each activation of the blood coagulation, i.e. with each damage to the tissue, be it of inflammatory, traumatic or degenerative genesis. The formation of fibrin as mediated by thrombin is basically a protective process with the purpose of quickly sealing any defects 10 caused to the vascular system. However, the formation of fibrin is also a pathogenic process. The appearance of a fibrin thrombus as the triggering cause of cardiac infarction is one of the most prominent problems in human medicine.

The role which fibrin plays during the extravastation of 15 inflammatory cells from the bloodstream into the tissue, which, on the one hand, is a desired process of the defense against pathogenic microorganisms or tumour cells occurring in the tissue, but, on the other hand, is a process which, by itself, induces or prolongues damage done to the tissue, has so 20 far not been examined at all or not to a sufficient extent. Fibrin binds to endothelial cells via its neo-N-terminus of Bbeta by means of the sequence to Bbeta and to cells in the bloodstream by means of the sequence Aalpha, thereby leading to the adhesion and transmigration of cells into the tissue.

The peptides or proteins according to the invention may prevent the adhesion of cells from the bloodstream to endothelial cells of the vascular wall and/or their subsequent transmigration from the blood into the tissue.

general formula II, wherein Z<sub>5</sub> denotes a peptide residue comprising the following amino acid sequence (SEQ ID NO

Asp Lys Lys Arg Glu Glu Ala Pro Ser Leu Arg Pro Ala Pro Pro Pro Ile Ser Gly Gly Gly Tyr Arq

Z<sub>1</sub> denotes a histidine residue, Arg denotes an arginine residue, Z<sub>3</sub> denotes a proline residue, Z<sub>4</sub> denotes a leucine residue,

prevents fibrin fragments from depositing on or adhering to 45 the vascular wall. Thus, it is rendered impossible that inflammatory cells are retained at the endothelial cells of the vascular walls of arteries and veins, and such cells are prevented from remaining at the vascular walls, thus being prevented from infiltrating the tissue any further.

A peptide or protein of the general formula II, wherein  $Z_5$ denotes a peptide residue comprising the following amino acid sequence (SEQ ID NO 292):

Glu Arg His Gln Ser Ala Cys Lys Asp Ser Asp Trp Pro Phe Cys Ser Asp Glu Asp Trp Asn Tyr Lys

and

 $Z_1$  denotes a proline residue, Arg denotes an arginine residue, Z<sub>3</sub> denotes a valine residue,  $Z_4$  denotes a valine residue,

has the effect of preventing the cells of the peripheral blood 65 from adhering to fibrin or fibrin fragments, hence prohibiting their migration in the tissue.

The described cleavage products are also known in the literature as peptide Bbeta and peptide Aalpha. Said above mentioned proadhesive and promigratory path is a completely new one for the system of controlling the migration of cells from the blood into the tissue. This function of the fibrin may be blocked by peptide Bbeta and also by peptide Aalpha.

Therefore, said peptides according to the invention are suitable as therapeutic agents for humans and animals in order to block the migration of cells from the blood into the tissue. Since fibrin or other fibrinogen products produced by proteolytic cleavage, such as, f.i., fibrinogen cleaved by an urokinase-plasminogen-activator, are generated only to a specific and regionally limited extent, i.e. at sites of inflammation, disturbed coagulation, arterial sclerosis, thrombosis and/or tumour growth, the effect of said therapeutic agent is regionally limited, which means that pathological side effects occurring in other places are not to be expected or only to a limited extent.

Preferable and completely unexpected fields of application for the peptides and/or proteins according to the invention consist in the preparation of pharmaceutical compositions for the therapy or prevention of local and/or generalized inflammations in the body in case of infectious genesis, based upon an auto-immune reaction, based upon a rheumatic disease, based upon a disorder in the immune system, based upon a genetic disease, for the prevention and/or therapy of the rejection occurring after organ transplants, of arterial sclerosis, of a reperfusion trauma, based upon arteriosclerotic and/or thrombotic diseases and an increased fibrin deposition. Such A peptide or protein according to the invention of the 30 a peptide, in particular Bbeta, is also excellently suitable for the preparation of a pharmaceutical composition which accomplishes the transportation of a further drug substance to human or animal endothelial cells. In doing so, the drug substance to be transported is coupled to the peptide at one 35 end and then, via VE-cadherin, deposits on a free spot of the vascular wall, i.e. on an endothelial cell.

> In the following, the invention is explained in further detail by way of examples.

#### **EXAMPLES**

#### Example 1

### Preparation of the Fibrinogen Cleavage Products

Non-polymerizing degradation products of fibrinogen were obtained by means of a decomposition involving cyanogen bromide according to Blombäck et al. (Nature 1968, 218; 130-134). The fibringen thus degraded largely consists of a 63 kD fragment, i.e. the N-terminal disulfide knot, NDSK, and comprises Aalpha-chain 1-51, Bbeta-chain 1-118 and gamma-chain 1-78. In order to obtain NDSK-II (NDSK minus fibrinopeptides A and B), the N-terminal amino acids of the Aalpha- and Bbeta-chains were cleaved off with throm-55 bin (20 units/1 μg NDSK) in three hours at room temperature and subsequently were treated with disopropylfluorophosphate in order to block thrombin activity. The NDSK-II thus obtained consisted of Aalpha-chain 17-51, Bbeta-chain 15-118 and gamma-chain 1-78.

In order to obtain NDSK-uPA, 500 µg of NDSK was treated with 200 units of urokinase-plasminogen-activator (uPA) of Messrs. Technoclone, Vienna, Austria, for one hour at 37° C. The reaction was stopped with 5 mM phenylmethylsulfonyl fluoride. The NDSK-uPA thus obtained is a NDSK and has no fibrinopeptide B.

As a negative control, a second fraction was obtained from the fibrinogen cleavage products referred to as FCB-2 accord-

ing to Nieuwenhuizen et al. (Biochem Biophys Acta 1983, 755; 531-533), which cleavage products were produced by being treated with cyanogen bromide. FCB-2 is a protein having a size of 43 kD and consists of Aalpha-chain 148-208, Bbeta-chain 191-305 and gamma-chain 95-265. For control purposes, thrombin and diisopropylfluorophosphate were added to said protein. That, however, did not result in any change to the protein (in the following, referred to as FCB-2-thr).

For the purpose of further negative controls, culture 10 medium (RPMI of Messrs. Life techn. Inc., Paisky, UK) was treated with thrombin as above and, subsequently, was inactivated (RPMI-thr) or was treated with uPA as above and was inactivated (RPMI-uPA).

#### Example 2

Peptide Aalpha (SEQ ID NO 293) corresponds to amino acids 1 to 28 of the alpha-chain of the fibrin and is identical to amino acids 17 to 45 of the Aalpha-chain of the fibrinogen:

Gly Pro Arg Val Val Glu Arg His Gln Ser Ala Cys Lys Asp Ser Asp Trp Pro Phe Cys Ser Asp Glu Asp Trp Asn Tyr Lys

Peptide Bbeta (SEQ IN NO 294) corresponds to amino acids 1 to 28 of the beta-chain of the fibrin, which is identical to amino acids 15 to 43 of the Bbeta-chain of the fibrinogen, which exhibits the following sequence:

Gly His Arg Pro Leu Asp Lys Lys Arg Glu Glu Ala Pro Ser Leu Arg Pro Ala Pro Pro Pro Ile Ser Gly Gly Gly Tyr Arg

By applying a fluorenylmethyloxycarbonyl (FMOC)-protective group strategy according to Carpino L. A. and Han. G Y, J. Amer. Chem. Soc. 1981; 37; 3404-3409, both peptides were synthesized by means of a solid-phase peptide synthesis according to Merrifield R. B., J. Amer. Chem. Soc. 1963; 85, 2149-2154, using a multiple peptide synthesizer. The crude peptides were purified by preparative reversed-phase HPLC via a Nucleosil 100-10, C18-column according to Engelhart 45 H. and Müller H. Chromatography 1984 19:77 as well as Henschen A., Hupe K. P. and Lottspeich F. High Performance Liquid Chromatography VCH 1985. As control peptides, peptides of the same length but comprising a randomized amino acid sequence were used.

#### Example 3

#### **HU-SCID Mouse-Model**

Human skin was transplanted onto the backs of SCID mice, and two weeks later human lymphocytes were injected into the peritoneum. The proceedings were according to Petzelbauer et al. (J. Invest. Dermatol. 1996, 107; 576-581). Then, fifteen mice thus prepared were injected in their tail veins with the following:

Further proce mm², 14+/-2

Examples 4 to inflammation.

- a) 100 µg of human NDSK-II
- b) 100 µg of human FCB-2
- c) 100 µg of peptide Aalpha
- d) 100 µg of peptide Bbeta
- e) 100 µg of randomized Aalpha
- f) 100 µg of randomized Bbeta

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Twenty-four hours later, the human skin was removed and the number of inflammatory sites, expressed in cells per 0.3 mm<sup>2</sup>, was evaluated and the mean value was determined with a standard deviation.

5 For a: 22+/-2.8

for b: 9+/-2.1

for c: 4+/-1.1

for d: 6+/-1.1

for e: 5+/-1.2

for f: 7+/-1.3

That allows the conclusion that NDSK-II causes inflammations, and hence said protein was used as a pathogenic substance. The other compounds per se do not exhibit any significant increase in the amount of inflammatory cells.

#### Comparative Example 4

Fifteen mice according to Example 3 were injected in their tail veins with

100 µg of human NDSK-II and

100 µg of randomized peptide Aalpha.

<sub>25</sub> Further proceedings were according to Example 3. Per 0.3 mm<sup>2</sup>, 23+/-3.5 inflammatory sites could be determined.

#### Comparative Example 5

Fifteen mice according to Example 3 were injected in their tail veins with

100 μg of human NDSK-II according to Example 1 and

100 μg of randomized peptide Bbeta.

Further proceedings were according to Example 3. Per 0.3 mm<sup>2</sup>, 24+/-2 inflammatory sites could be determined.

#### Example 6

Fifteen mice according to Example 3 were injected with

100 µg of human NDSK-II and

100 μg of synthesized peptide Aalpha.

Further proceedings were according to Example 3. Per 0.3 mm<sup>2</sup>, 21+/-2.2 inflammatory sites could be determined.

### Example 7

Fifteen mice according to Example 3 were injected in their tail veins with

100 μg of human NDSK-II and

55 100 μg of synthesized peptide Bbeta.

Further proceedings were according to Example 3. Per 0.3 mm<sup>2</sup>, 14+/-2 inflammatory sites could be determined.

Examples 4 to 7 show that peptide Bbeta blocks lymphocytic inflammation.

#### Comparative Example 8

Endothelial cells from human umbilical veins (HUVEC)

65 were marked with a red fluorescent dye (Cell Tracker Orange,

1 μl/ml, Molecular Probes, Eugene, Oreg.) and were dispersed on a collagen matrix (Collaborative Biomedical Prod-

**8** Example 10

ings were in accordance with Example 8.

ucts, Bedford, Mass.). Upon confluence of the endothelial cells, peripheral mononuclear blood cells (PBMC) ( $10^5$  cells per 25 mm²) marked with a green fluorescent dye (Cell Tracker Green, 1  $\mu$ /ml, Molecular Probes of Messrs. Eugene, Oreg.) were superimposed. Thereafter, the cells were incubated at  $37^{\circ}$ C. for twelve hours.

Adhering cells that had transmigrated into the gel were photographed with a laser-scan microscope, were converted into pixels and were evaluated by means of an 'NIH image" according to Gröger et al. (J. Immunol. Method 1999; 222: 101-109).

It was feasible to determine the number of adherent cells per 0.1 mm<sup>2</sup> such as mentioned under "adhesion." It was feasible to determine the number of migrated cells per 0.04 mm<sup>3</sup> such as mentioned under "migration." The mean value of three times three trials was evaluated together with the standard deviation.

		adhesion	migration
a) RPMI-uPA	0.1 μg/ml	40 +/- 4	4 +/- 3
,	1.0 μg/ml	38 +/- 2	5 +/- 2
	10.0 μg/ml	32 +/- 4	5 +/- 1
b) NDSK	0.1 μg/ml	31 +/- 18	6 +/- 3
•	1.0 μg/ml	35 +/- 18	5 +/- 2
	10.0 μg/ml	36 +/- 24	6 +/- 3
c) NDSK-II	0.1 μg/ml	55 +/- 21	12 +/- 5
,	1.0 μg/ml	67 +/- 31	19 +/- 12
	10.0 μg/ml	65 +/- 31	19 +/- 10
d) NDSK-uPA	0.1 μg/ml	58 +/- 3	10 +/- 2
•	1.0 μg/ml	60 +/- 3.5	14 +/- 3
	10.0 μg/ml	65 +/- 3	18 +/- 1.5
e) FCB2	0.1 μg/ml	30 +/~ 26	6 +/- 4
	1.0 μg/ml	10 +/- 10	3 +/- 2
	10.0 μg/ml	21 +/- 7	5 +/- 4
f) FCB-2-thr	0.1 μg/ml	20 +/- 12	6 +/- 5
	1.0 μg/ml	23 +/- 13	7 +/- 5
	10.0 μg/ml	26 +/- 11	4 +/- 2
g) RPMI-thr	0.1 μg/ml	29 +/- 15	4 +/- 5
	1.0 μg/ml	26 +/- 14	5 +/- 5
	10.0 μg/ml	41 +/- 20	5 +/- 4

That allows the conclusion that NDSK-II results in significant migrations of peripheral blood-monocellular cells (PBMC) to a greater extent than NDSK-uPA and hence exhibits pathogenic activity. None of the controls a), b), e), f) and g) resulted in any significant migration.

#### Example 9

100 µg of NDSK-II and Bbeta or Bbeta randomized were added to the collagen matrix according to Example 8 comprising the suspension of PBMC, and further proceedings were in accordance with Example 8.

	adhesion	migration
a) no addition of NDSK-II	38 +/- 15	6 +/- 4
b) only 100 μg of NDSK-II	73 +/- 29	16 +/- 7
c) 10 µg of Bbeta + NDSK-II	63 +/- 33	7 +/- 4
d) 100 μg of Bbeta + NDSK-II	47 +/- 34	5 +/- 4
e) 1000 µg of Bbeta + NDSK-II	52 +/- 27	10 +/- 6
f) 10 μg of Bbeta randomized + NDSK-II	77 +/- 33	16 +/- 6
g) 100 µg of Bbeta randomized + NDSK-II	86 +/- 35	15 +/- 6
n) 1000 µg of Bbeta randomized + NDSK-II	78 +/- 31	13 +/- 8

As can be gathered from those test results, peptide Bbeta blocks inflammations.

 $100~\mu g$  of NDSK-II and Aalpha or Aalpha randomized were added to the collagen matrix according to Example 8 comprising the suspension of PBMC, and further proceed-

	adhesion	migration
a) no addition of NDSK-II b) only NDSK-II c) 10 µg of Aalpha + NDSK-II d) 100 µg of Aalpha + NDSK-II 5 e) 1000 µg of Aalpha + NDSK-II f) 10 µg of Aalpha randomized + NDSK-II g) 100 µg of Aalpha randomized + NDSK-II h) 1000 µg of Aalpha randomized + NDSK-II	42 +/- 6 96 +/- 11 69 +/- 12 73 +/- 13 70 +/- 6 70 +/- 6 65 +/- 16 70 +/- 12	10 +/- 1 24 +/- 3 21 +/- 4 15 +/- 6 13 +/- 5 25 +/- 2 24 +/- 3 26 +/- 3

As can be gathered from the test results, peptide Aalpha blocks the migration of PBMC only partially.

#### Example 11

Since PBMC substantially consists of a mixture of lymphocytes and monocytes, pure lymphocytes instead of PBMC (as in Examples 8-10) were used in Example 11.

100 µg of NDSK-uPA or 100 µg of NDSK-II, respectively,
 and Aalpha or Bbeta, respectively, were added to the collagen matrix according to Example 8 comprising endothelial cells and lymphocytes.

33		adhesion	migration
	a) no addition	68 +/- 8	16 +/- 3
	b) NDSK-uPA	143 +/- 11	53 +/- 5
	c) NDSK-II	119 +/- 11	43 +/- 4
40	d) only 100 µg of Bbeta	58 +/- 18	14 +/- 1
40	e) NDSK-uPA + 100 µg of Bbeta	74 +/- 8	19 +/- 2
	f) NDSK-II + 100 µg of Bbeta	74 +/- 8	17 +/- 3
	g) only 100 µg of Aalpha	77 +/- 4	18 +/- 1
	h) NDSK-uPA + 100 µg of Aalpha	131 +/- 4	40 +/- 3
	i) NDSK-II + 100 µg of Aalpha	131 +/- 4	44 +/- 4
	j) only 100 µg of Bbeta randomized	75 +/- 5	19 +/- 1
45	k) NDSK-uPA + 100 µg of Bbeta randomized	134 +/- 13	46 +/- 4
	l) NDSK-II + 100 µg of Bbeta randomized	120 +/- 12	42 +/- 4

Those test results show

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- that both NDSK-II and NDSK-uPA promote lymphocytic inflammation,
- 2) that peptide Bbeta completely blocks the lymphocytic adhesion and migration induced by NDSK-II and NDSK-uPA, whereas peptide Aalpha exhibits no blocking activity, which suggests that the free alpha-chain is not required for inducing the adhesion and migration of the lymphocytes.

#### Example 12

The proceedings were in accordance with Example 11, except for pure monocytes being used instead of lymphocytes. 100 µg of NDSK-uPA or 100 µg of NDSK-II, respectively, was added to peptide Aalpha, randomized Aalpha, Bbeta or randomized Bbeta.

	adhesion	migration
a) no addition	43 +/- 8	7 +/- 1
b) NDSK-uPA	48 +/- 10	10 +/- 2
c) NDSK-II	90 +/- 11	19 +/- 6
d) 100 µg of Bbeta	59 +/- 7	5 <b>+</b> /- 1
e) NDSK-uPA + 100 µg of Bbeta	61 +/- 11	8 +/- 3
f) NDSK-II + 100 µg of Bbeta	70 +/- 7	7 +/- 5
g) 100 µg of Bbeta randomized	40 +/- 7	6 +/- 1
h) NDSK-uPA + 100 µg of Bbeta randomized	45 +/- 5	8 +/- 3
g) NDSK-II + 100 µg of Bbeta randomized	92 +/- 10	20 +/- 7
j) 100 μg of Aalpha	59 +/- 6	5 +/- 1
k) NDSK-uPA + 100 µg of Aalpha	62 +/- 4	8 +/- 5
i) NDSK-II + 100 µg of Aalpha	68 +/- 10	9 +/- 6
m) 100 µg of Aalpha randomized	58 +/- 7	6 +/- 1
n) NDSK-uPA + 100 µg of Aalpha randomized	50 +/- 10	10 +/- 4
o) NDSK-II + 100 μg of Aalpha randomized	108 +/- 8	21 +/- 5

Those test results show that only NDSK-II and not NDSK-uPA promotes the migration of monocytes, which means that both the alpha-chain and the beta-chain have to exhibit a free N-terminal end and block the migration of the monocytes.

#### Example 13

The proceedings were in accordance with Example 11, with  $_{30}$  pure lymphocytes being used. 100 µg of NDSK-uPA or 100 µg of NDSK-II, respectively, was added to the short peptide salts derived from Aalpha Gly Pro Arg (Pro)—NH $_2$  acetate (Aalpha derivative) or derived from Bbeta Gly His Arg Pro-OH acetate (Bbeta derivative).

	adhesion	migration
a) no addition	60 +/~ 8	14 +/- 1
b) NDSK-uPA	149 +/- 12	57 +/- 5
c) NDSK-II	121 +/- 11	48 +/- 7
d) only 100 μg of Bbeta derivative	58 +/~ 10	12 +/- 9
e) NDSK-uPA + 100 µg of Bbeta derivative	70 +/~ 8	16 +/- 3
f) NDSK-II + 100 µg of Bbeta derivative	69 +/- 7	14 +/- 5
g) only 100 μg of Aalpha derivative	77 +/~ 4	18 +/- 1
h) NDSK-uPA + 100 μg of Aalpha derivative	134 +/~ 4	48 +/- 5
i) NDSK-II + 100 μg of Aalpha derivative	131 +/~ 7	49 +/- 6
j) only 100 μg of Bbeta derivative randomized	70 +/~ 5	14 +/- 7
k) NDSK-uPA + 100 μg of Bbeta derivative randomized	130 +/~ 12	49 +/- 6
<ol> <li>NDSK-II + 100 μg of Bbeta derivative randomized</li> </ol>	120 +/~ 10	55 +/- 8

Said experiment allows the conclusion that, if lymphocytic migration is inhibited, those short peptides, added continuously in an appropriate manner, exhibit the same activity as do the long peptides.

#### Example 14

The proceedings were in accordance with Example 12, with pure monocytes being used. 100 mg of NDSK-uPA or 100 µg of NDSK-II, respectively, was added to the short peptide salts Aalpha Gly Pro Arg (Pro)—NH<sub>2</sub> acetate (Aalpha derivative) or Bbeta Gly His Arg Pro-OH acetate (Bbeta derivative).

		adhesion	migration
5	a) no addition	40 +/- 8	5 +/- 1
	b) NDSK-uPA	54 +/ 9	7 +/- 2
	c) NDSK-II	85 +/- 11	22 +/- 6
	d) 100 µg of Bbeta derivative	52 +/- 7	6 +/- 1
	e) NDSK-uPA + 100 µg of Bbeta derivative	61 +/- 11	8 +/- 3
	f) NDSK-II + 100 µg of Bbeta derivative	68 +/- 7	8 +/- 4
10	g) 100 µg of Bbeta derivative randomized	40 +/- 7	6 +/- 1
•	h) NDSK-uPA + 100 µg of Bbeta derivative	44 +/- 6	8 +/- 2
	randomized i) NDSK-II + 100 μg of Bbeta derivative	92 +/- 10	23 +/- 7
	randomized	50 / 5	
	j) 100 μg of Aalpha derivative	50 +/- 5	
15	<ul> <li>k) NDSK-uPA + 100 μg of Aalpha derivative</li> </ul>	60 +/- 5	7 +/- 6
	<ol> <li>NDSK-II + 100 µg of Aalpha derivative</li> </ol>	64 +/- 11	8 +/- 2
	<ul> <li>m) 100 μg of Aalpha derivative randomized</li> </ul>	54 +/- 10	6 +/- 3
	n) NDSK-uPA + 100 μg of Aalpha derivative randomized	50 +/- 10	10 +/- 4
20	o) NDSK-II + 100 µg of Aalpha derivative	99 +/- 8	21 +/- 7

Said experiment allows the conclusion that, if monocytic migration is inhibited, those short peptides, added continuously in an appropriate manner, exhibit the same activity as do the long peptides.

#### Example 15

The tests were carried out on male wistar rats weighing between 220 g and 280 g. The rats were given standard food and water. For carrying out the test, the rats were anaesthetized and artifically respirated with a frequency of 70 pulses per minute, whereby from 8 ml to 10 ml per kilogram of a gas containing 30 % by volume of oxygen and having an overpressure of from 1 mm to 2 mm mercury was emitted. The cardiac artery on the right hand side was equipped with a measuring cannula, and the blood pressure in the artery as well as the heartbeats were determined. The pressure rate was determined as a product of the blood pressure in the artery and of the heartbeat rate with the dimension mm mercury/minute/ 10<sup>3</sup>. The vein on the right hand side was equipped with a measuring cannula for doping the test substances. After carrying out the surgical treatment, 2 ml of rat blood was supplied to the heart. Thirty minutes later, the cardiac artery on the left hand side was occluded. Another twenty-five minutes later, the occlusion was released in order to resupply the ischaemic area with blood. At that point of time, 800 µg/kg of peptide Bbeta or peptide Bbeta randomized, respectively, was intravenously administered to half of the animals, and then two hours were allowed to pass.

In order to distinguish between damaged and undamaged cardiac tissue, the cardiac artery on the left hand side was then supplied with evans blue dye at a concentration of 2% by weight. Thereupon, the removed heart was dissected by five horizontal cuts, the right hand wall of the vein was removed and the sections were treated with triphenyltetratolchloride (1% by weight) for twenty minutes at 37° C. so as to be able to distinguish between normal tissue and infarct tissue. The sections were evaluated by computer-sustained planimetry.

Because of the vascular occlusion, 62.5% of the cardiac muscle in the hearts of the reference rats was threatened, as opposed to 60% in the hearts of the test rats. In the hearts of the reference rats, 46% of the endangered tissue was dead, as opposed to 29% in the hearts of the test rats. That corresponds to a 37% reduction of dead tissue (p<0.05).

The substances according to the invention as well as the use of the substances according to the invention for preparing a pharmaceutical composition are of special significance:

For a pharmaceutical composition used in the therapy of diseases caused by the tissue-damaging effect of autoreactive 5

Among those are diseases fitting into the sphere of autoimmunity, such as collagenoses, rheumatic diseases, psoriasis and post-/parainfectious diseases and diseases caused by a graft versus host reaction. A healing effect occurs, since said pharmaceutical composition blocks the migration of lymphocytes into the tissue. Thus, the lymphocytes remain in the bloodstream and are incapable of producing an autoreactive tissue-damaging effect.

A healing effect occurs with a drug for the therapy and/or 15 prevention of the rejection occurring after organ transplants, since said drug prevents the migration of lymphocytes from the bloodstream into the foreign organ and hence the foreign organ cannot be destroyed by autoreactive lymphocytes.

A healing effect occurs with a drug for the therapy and/or 20 prevention of arterial sclerosis after organ transplants, since said drug prohibits the migration of lymphocytes and monocytes into the vascular wall and hence prevents the activation of the cells of the vascular wall. In doing so, the occurrence of prevented.

A healing effect occurs with a drug for the therapy and/or prevention of a reperfusion trauma following a surgically or pharmaceutically induced restoration of the blood flow such as, f.i. after cardiac infarction, apoplectic stroke, after vascu- 30 lar surgery, bypass surgery and organ transplants, since said

12

drug inhibits the migration of lymphocytes and monocytes into the vascular wall. The reperfusion trauma is caused by oxygen deficiency/acidosis occurring in the cells of the vessel during the restoration of the blood flow and leads to their activation. Thereby, lymphocytes and monocytes adhere to the vascular wall and migrate into the same. The fact that lymphocytes and monocytes are prevented from adhering to and migrating into the vascular wall brings about a decrease in the hypoxia/acidosis-induced damage, without any permanent vascular damage being caused by the subsequent inflammatory reaction.

A healing effect occurs with a drug for the therapy and/or prevention of arterial sclerosis following metabolic diseases or ageing processes, since said drug inhibits the migration of lymphocytes and monocytes into the vascular wall and hence inhibits the progredience of the arteriosclerotic plaque resulting therefrom.

The pharmaceutical composition according to the invention may also be used for transporting a further drug substance. The pharmaceutical composition according to the invention specifically binds a surface molecule to endothelial cells. Thus, drug substances coupled thereto may be contacted with endothelial cells at high concentrations, without them being able to trigger side reactions in other places. The arterial sclerosis following organ transplants is minimized or 25 use of substances inhibiting cell division may be mentioned as an example, which substances may exhibit an antiangiogenetic effect after having been adducted specifically to endothelial cells. In that case, tumour patients experience a healing effect, since the growth of the tumour is blocked by preventing the proliferation of endothelial cells and hence by avoiding neoangiogenesis.

TABLE 1

	Peptides of Formula I: Gly - His/Pro - Arq - Xaa, - Xaa,	
SEQUENCE		SEQ ID NO
Gly His Arg		1
Gly Pro Arg		2
Gly His Arg Xa	ua	3
Gly Pro Arg Xa	ua	4
Gly His Arg Xa	a Xaa	5
Gly Pro Arg Xa	a Xaa	6
Gly His Arg Xa	a Xaa Xaa	7
Gly Pro Arg Xa	a Xaa Xaa	8
Gly His Arg Xa	a Xaa Xaa Xaa	9
Gly Pro Arg Xa	a Xaa Xaa Xaa	10
Gly His Arg Xa	a Xaa Xaa Xaa	11
Gly Pro Arg Xa	a Xaa Xaa Xaa	12
Gly His Arg Xa	a Xaa Xaa Xaa Xaa	13
Gly Pro Arg Xa	a Xaa Xaa Xaa Xaa	14
Gly His Arg Xa	a Xaa Xaa Xaa Xaa Xaa	15
Gly Pro Arg Xa	a Xaa Xaa Xaa Xaa Xaa	16
Gly His Arg Xa	a Xaa Xaa Xaa Xaa Xaa Xaa	17
Gly Pro Arg Xa	a Xaa Xaa Xaa Xaa Xaa Xaa	18

### TABLE 1-continued

			Pe	ept ic	des_c	of Fo			: GJ2			_		X	aa <sub>2</sub>	- Xa	a <sub>20</sub>			
SEQU	ENC	2		_		_												SEQ	ID	NO
Gly	His	Arq	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa								19	
Gly	Pro	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa								20	
Gly	His	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa							21	
Gly	Pro	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa							22	
Gly	His	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa						23	
Gly	Pro	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa						24	
Gly	His	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa					25	
Gly	Pro	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa					26	
Gly	His	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				27	
Gly	Pro	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				28	
Gly		_																	29	
Gly																	.,		30	
Gly		_																	31	
Gly		_			хаа Хаа														32	
Xaa		arg	Aaa	naa	Ada	Aud	Aqu	Add	лаа	naa	Ada	Add	Adu	Add	Add	naa	naa		J J	
Gly Xaa		Arg	Xaa	Xaa	Xaa	Xaa	Хаа	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Хаа	Xaa	Xaa	Xaa		34	
Gly Xaa	His	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	:	295	
Gly Xaa	Pro	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	:	296	
Gly Xaa		_	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa		35	
Gly Xaa		_	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa		36	
Gly Xaa				Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa		37	
Gly Xaa					Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa		38	
Gly Xaa						Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa		39	
Gly Xaa						Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa		40	
Gly	His	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa		41	
Xaa :	Xaa	Xaa	Xaa	Xaa	Xaa															
Gly Xaa						Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa		42	
Gly Xaa	His Xaa	Arg Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa		43	
Gly Xaa							Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Хаа	Xaa		44	
Gly : Xaa :	His Xaa	Arg Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Хаа	Xaa		45	

TABLE 1-continued

			Pe	ptic	les c	of Fo	ormu]	la I	Gly	7 - I	lis/E	ro -	Arc	1 - >	⟨aa₂	- Xa	a <sub>20</sub>	
SEQ	JENC!	3																SEQ ID NO
-		_			Xaa Xaa			Xaa	Хаа	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	46
		_			Xaa Xaa			Xaa Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	47
		_			Xaa Xaa			Xaa Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	48
_		_						Xaa Xaa		Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	49
		-						Xaa Xaa		Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	50
		-						Xaa Xaa			Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	51
_		_						Xaa Xaa			Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	52
_		_						Xaa Xaa				Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	53
		_						Xaa Xaa				Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	54
		_						Xaa Xaa					Xaa	Xaa	Xaa	Xaa	Xaa	55
		_						Xaa Xaa					Xaa	Xaa	Xaa	Xaa	Xaa	56
								Xaa Xaa						Xaa	Xaa	Xaa	Xaa	57
Gly	Pro	Arg	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	58

TABLE 2

			P —	epti	des	of F	ormula II: Gly - His/Pro - Arg - Pro/Val - Leu/Val - Xaa <sub>2</sub> - Xaa <sub>10</sub>		
SEQ	UENC	Ε						SEQ	ID NO
Gly	His	Arg	Pro	Leu	Xaa	Xaa			59
Gly	Pro	Arg	Pro	Leu	Xaa	Xaa			60
Gly	His	Arg	Val	Leu	Xaa	Xaa			61
Gly	Pro	Arg	Val	Leu	Xaa	Xaa			62
Gly	His	Arg	Pro	Val	Xaa	Xaa			63
Gly	Pro	Arg	Pro	Val	Xaa	Xaa			64
Gly	His	Arg	Val	Val	Xaa	Xaa			65
Gly	Pro	Arg	Val	Val	Xaa	Xaa		•	66
Gly	His	Arg	Pro	Leu	Xaa	Xaa	Xaa	6	67
Gly	Pro	Arg	Pro	Leu	Xaa	Xaa	Xaa	6	68
Gly	His	Arg	Val	Leu	Xaa	Xaa	Xaa	é	59
Gly	Pro	Arg	Val	Leu	Xaa	Xaa	Xaa	7	70
Gly	His	Arg	Pro	Val	Xaa	Xaa	Xaa	7	71

TABLE 2-continued

Peptides of Formula II: Gly - His/Pro - Arg - Pro/Val - Leu/Val - Xaa <sub>2</sub> - Xaa <sub>30</sub>	
SEQUENCE	SEQ ID NO
Gly Pro Arg Pro Val Xaa Xaa Xaa	72
Gly His Arg Val Val Xaa Xaa Xaa	73
Gly Pro Arg Val Val Xaa Xaa Xaa	74
Gly His Arg Pro Leu Xaa Xaa Xaa Xaa	75
Gly Pro Arg Pro Leu Xaa Xaa Xaa Xaa	76
Gly His Arg Val Leu Xaa Xaa Xaa Xaa	77
Gly Pro Arg Val Leu Xaa Xaa Xaa Xaa	78
Gly His Arg Pro Val Xaa Xaa Xaa Xaa	79
Gly Pro Arg Pro Val Xaa Xaa Xaa Xaa	80
Gly His Arg Val Val Xaa Xaa Xaa Xaa	81
Gly Pro Arg Val Val Xaa Xaa Xaa	82
Gly His Arg Pro Leu Xaa Xaa Xaa Xaa Xaa	83
Gly Pro Arg Pro Leu Xaa Xaa Xaa Xaa	84
Gly His Arg Val Leu Xaa Xaa Xaa Xaa Xaa	85
Gly Pro Arg Val Leu Xaa Xaa Xaa Xaa Xaa	86
Gly His Arg Pro Val Xaa Xaa Xaa Xaa	87
Gly Pro Arg Pro Val Xaa Xaa Xaa Xaa	88
Gly His Arg Val Val Xaa Xaa Xaa Xaa	89
Gly Pro Arg Val Val Xaa Xa.a Xaa Xaa Xaa	90
Gly His Arg Pro Leu Xaa Xaa Xaa Xaa Xaa Xaa	91
Gly Pro Arg Pro Leu Xaa Xaa Xaa Xaa Xaa Xaa	92
Gly His Arg Val Leu Xaa Xaa Xaa Xaa Xaa Xaa	93
Gly Pro Arg Val Leu Xaa Xaa Xaa Xaa Xaa Xaa	94 95
Gly His Arg Pro Val Xaa Xaa Xaa Xaa Xaa Xaa	96
Gly Pro Arg Pro Val Xaa Xaa Xaa Xaa Xaa Xaa Caa Caa Caa Caa	97
Gly Pro Arg Val Val Xaa Xaa Xaa Xaa Xaa Xaa	98
Gly His Arg Pro Leu Xaa Xaa Xaa Xaa Xaa Xaa	99
Gly Pro Arg Pro Leu Xaa Xaa Xaa Xaa Xaa Xaa	100
Gly His Arg Val Leu Xaa Xaa Xaa Xaa Xaa Xaa	101
Gly Pro Arg Val Leu Xaa Xaa Xaa Xaa Xaa Xaa	102
Gly His Arg Pro Val Xaa Xaa Xaa Xaa Xaa Xaa Xaa	103
Gly Pro Arg Pro Val Xaa Xaa Xaa Xaa Xaa Xaa	104
Gly His Arg Val Val Xaa Xaa Xaa Xaa Xaa Xaa	105
Gly Pro Arg Val Val Xaa Xaa Xaa Xaa Xaa Xaa	106
Gly His Arg Pro Leu Xaa Xaa Xaa Xaa Xaa Xaa Xaa Xaa	107
Gly Pro Arg Pro Leu Xaa Xaa Xaa Xaa Xaa Xaa Xaa Xaa	108

TABLE 2-continued

			P	epti	des d	of Fo	_				His			:g -	Pro	Val -		
						_					a <sub>2</sub> -						CEO	ID NO
	ENCE											**						10 100
-		-									Xaa							
-											Xaa							110
Gly	His	Arg	Pro	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa						111
Gly	Pro	Arg	Pro	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa						112
Gly	His	Arg	Val	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa						113
Gly	Pro	Arg	Val	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa					:	114
Gly	His	Arg	Pro	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				:	115
Gly	Pro	Arg	Pro	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				:	116
Gly	His	Arg	Val	Leu	Xaa	Xaa	Хаа	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				:	117
Gly	Pro	Arg	Val	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa					118
Gly	His	Arg	Pro	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				:	119
Gly	Pro	Arg	Pro	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				:	120
Gly	His	Arg	Val	Val	Xaa	Xaa	Хаа	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				:	121
Gly	Pro	Arg	Val	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				:	122
Gly	His	Arg	Pro	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa			:	123
Gly	Pro	Arg	Pro	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				124
Gly	His	Arg	Val	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa			:	125
Gly	Pro	Arg	Val	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				126
Gly	His	Arg	Pro	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				127
Gly	Pro	Arg	Pro	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				128
Gly	His	Arg	Val	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				129
Gly	Pro	Arg	Val	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa				130
											Xaa				Xaa			131
-		_									Xaa							132
•		_									Xaa							133
-		_									Xaa							134
		_									Xaa							135
_		_									Xaa							136
		_									Хаа							137
		_									Xaa							
·		_														V		138
-		-									Xaa							139
											. Хаа							140
											Xaa							141
											Xaa							142
											Xaa							143
											Xaa							144
Gly	His	Arg	Val	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa		145

TABLE 2-continued

Gly Pro Arg Gly His Arg Gly His Arg Gly Pro Arg Xaa  Gly Pro Arg Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa	Pro Pro Val Val Pro Val Pro Val Val Pro Val	Leu Leu Val Val Val Leu Leu Leu Leu	Xaaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa	SEQ ID NO  146  147  148  149  150  151  152  153							
Gly His Arg Gly Pro Arg Kaa Arg	Pro Pro Val Val Pro Val Pro Val Val Pro Val	Leu Leu Val Val Val Leu Leu Leu Leu	Xaaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa	147 148 149 150 151							
Gly Pro Arg Gly His Arg Gly His Arg Gly His Arg Gly Pro Arg Gly His Arg Gly Pro Arg Gly His Arg Xaa Gly Pro Arg Xaa Gly Pro Arg Xaa Gly Pro Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly His Arg Xaa Gly His Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly His Arg Xaa	Pro Val Pro Val Val Pro Val Val Val Val	Leu Leu Val Val Val Leu Leu Leu	Xaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	148 149 150 151 152						
Gly His Arg Gly Pro Arg Gly Pro Arg Gly His Arg Gly Pro Arg Gly His Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly His Arg Xaa	Vall Pro Vall Pro Vall Vall Vall Vall Vall Vall	Leu Val Val Val Leu Leu Leu	Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	149 150 151 152
Gly Pro Arg Gly His Arg Gly His Arg Gly His Arg Gly Pro Arg Kaa Arg Gly His Arg Kaa Arg Gly His Arg Kaa Arg Gly Pro Arg Kaa Arg Gly Pro Arg Kaa Arg	Val Pro Val Pro Pro Val Val Val	Leu Val Val Val Leu Leu	Xaa Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa	150 151 152						
Gly His Arg Gly Pro Arg Gly His Arg Gly Pro Arg Cly His Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly His Arg Xaa Gly His Arg Xaa	Pro Val Val Pro Val Val	Val Val Val Leu Leu	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa	Xaa Xaa Xaa	151 152						
Gly Pro Arg Gly His Arg Gly His Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly His Arg Xaa Gly His Arg Xaa Gly Pro Arg Xaa	Pro Val Val Pro Val	Val Val Leu Leu	Xaa Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa	Xaa Xaa Xaa Xaa	Xaa Xaa Xaa	Xaa Xaa Xaa	Xaa Xaa Xaa	Xaa Xaa	152						
Gly His Arg Gly Pro Arg Kaa Gly Pro Arg Kaa Gly His Arg Kaa Gly Pro Arg Kaa Gly His Arg Kaa Gly His Arg Kaa Gly His Arg Kaa Gly Pro Arg Kaa	Val Val Pro Pro Val	Val Val Leu Leu	Xaa Xaa Xaa Xaa	Xaa Xaa Xaa	Xaa Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa							
Gly Pro Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly His Arg Xaa Gly His Arg Xaa Gly Pro Arg Xaa Gly His Arg	Val Pro Pro Val	Val Leu Leu	Xaa Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa	Xaa	Xaa								153
Gly His Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly Pro Arg Xaa Gly Pro Arg Xaa Gly His Arg Xaa Gly His Arg Xaa Gly His Arg Xaa Gly Pro Arg Xaa	Pro Pro Val	Leu Leu Leu	Xaa Xaa	Xaa	Xaa				Xaa	Xaa	v		y	Yan	Xaa	
Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly His Arg Xaa  Gly His Arg Xaa  Gly His Arg	Pro Val	Leu Leu	Xaa			Xaa	Xaa	Xaa			хаа	Xaa	Add	nad		154
Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly Pro Arg Xaa  Gly Pro Arg Xaa  Gly His Arg	Val Val	Leu		Xaa	Xaa				Xaa	Xaa	Xaa	Xaa	Xaa	Хаа	Xaa	155
Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly His Arg	Val		Xaa			Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Хаа	Xaa	156
Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly His Arg		Leu		Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	157
Xaa  Gly Pro Arg Xaa  Gly His Arg Xaa  Gly Pro Arg Xaa  Gly His Arg	Pro		Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	158
Xaa Gly His Arg Xaa Gly Pro Arg Xaa Gly His Arg		Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	159
Xaa Gly Pro Arg Xaa Gly His Arg	Pro	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	160
Xaa Gly His Arg	Val	Val	Xaa	Xaa	Xaa	Хаа	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	161
	Val	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	162
	Pro	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	163
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Gly His Arg Xaa Xaa	Val	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	165
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Gly His Arg Xaa Xaa Xaa	Pro	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	171
Gly Pro Arg Xaa Xaa Xaa	Pro	Leu	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	172
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TABLE 2-continued

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	Pro Xaa			Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	176
-	His Xaa	_		Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	177
	Pro Xaa			Val	Xaa	Xaa	Xaa	Хаа	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Хаа	178
	His Xaa	_			. Xaa	Xaa	Xaa	Xaa	Xaa	Хаа	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	179
	Pro Xaa				Xaa	Xaa	Xaa	. Xaa	Xaa	Xaa	Xaa	Xaa	. Xaa	Xaa	Хаа	Xaa	Xaa	180
	His Xaa				. Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	181
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	His Xaa				Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	183
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	His Xaa			Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	185
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TABLE 2-continued

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Хаа	Xaa	Xaa	Xaa	Xaa	Xaa Xaa	Xaa	Xaa	Xaa										220
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TABLE 2-continued

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TABLE 2-continued

			I	epti	des	of F	ormu					s/Pro		rg -	Pro	o/Val	l -	
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-		_			Xaa Xaa								Xaa	Xaa	Xaa	ı Xaa	. Xaa	249
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Gly Xaa	Pro Xaa	Arg Xaa	Pro Xaa	Leu Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa Xaa	Xaa	Xaa	Xaa	268
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TABLE 2-continued

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Gly	Pro	Arg	Val	Val	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	Xaa	290
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Xaa Xaa Xaa

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Xaa Xaa Xaa
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Ile Ser Gly Gly Gly Tyr Arg
<210> SEQ ID NO 292
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<210> SEQ ID NO 293
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<212> TYPE: PRT
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Trp Pro Phe Cys Ser Asp Glu Asp Trp Asn Tyr Lys
<210> SEQ ID NO 294
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<212> TYPE: PRT
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Pro Ala Pro Pro Ile Ser Gly Gly Gly Tyr Arg
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Xaa Xaa Xaa
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The invention claimed is:

1. A method of treating rejection of a transplanted tissue in subject comprising administering to the subject a peptide

(SEQ ID NO:294) Gly His Arg Pro Leu Asp Lys Lys Arg Glu Glu Ala 60

Pro Ser Leu Arg Pro Ala Pro Pro Pro Ile Ser Gly

Gly Gly Tyr Arg

or a salt or amide thereof, in an amount effective to treat 65 rejection of transplanted tissue, wherein the amino terminus is



wherein R1 and R2 are either the same or different, wherein R1 and R2 are each selected from the group consisting of hydrogen and a saturated or unsaturated hydrocarbon residue, said residue having from 1 to 10 carbon atoms, and

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wherein the peptide, or a salt or amide thereof, comprises a peptide derived from a source selected from the group consisting of the Aalpha-chain of fibrin and the Bbeta chain of fibrin.

2. A method of inhibiting inflammation of a transplanted 5 tissue in a subject comprising administering to the subject a peptide

(SEQ ID NO:294) 10

Gly His Arg Pro Leu Asp Lys Lys Arg Glu Glu Ala

Pro Ser Leu Arg Pro Ala Pro Pro Pro Ile Ser Gly

Gly Gly Tyr Arg

or a salt or amide thereof, in an amount effective to treat rejection of transplanted tissue, wherein the amino terminus is 176



wherein R1 and R2 are either the same or different,

wherein R1 and R2 are each selected from the group consisting of hydrogen and a saturated or unsaturated hydrocarbon residue, said residue having from 1 to 10 carbon atoms.

3. The method of claim 2, wherein the peptide, or a salt or amide thereof, comprises a peptide derived from a source selected from the group consisting of the Aalpha-chain of fibrin and the Bbeta chain of fibrin.

\* \* \* \* \*